

SYSTEM:OS - DIALOG OneSearch

File 155:MEDLINE(R) 1950-2006/Dec 16

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*File 155: MEDLINE has resumed updating with UD20061209. Please see HELP NEWS 154 for details.

File 55:Biosis Previews(R) 1993-2007/Jan W3

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File 34:SciSearch(R) Cited Ref Sci 1990-2007/Jan W3

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File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec

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File 340:CLAIMS(R)/US Patent 1950-07/Jan 25

(c) 2007 IFI/CLAIMS(R)

*File 340: The 2006 reload is online as of December 1, 2006.

IPCR/8 is available.

Set	Items	Description
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? s prb(2n)bind?		
	10062	PRB
	2392327	BIND?
S1	921	PRB(2N)BIND?
? s casein(w)kinase(w)ii(5n)phosphoryl?		
	70955	CASEIN
	866055	KINASE
	2053110	II
	504936	PHOSPHORYL?
S2	2739	CASEIN(W) KINASE(W) II (5N) PHOSPHORYL?
? s s1 and s2		
	921	S1
	2739	S2
S3	26	S1 AND S2
? s (second or two) (5n) casein		
Processing		
	3532539	SECOND
	7531858	TWO
	70955	CASEIN
S4	1476	(SECOND OR TWO) (5N) CASEIN
? s s3 and s4		
	26	S3
	1476	S4
S5	1	S3 AND S4
? t s5/3,k,ab/1		

5/3,K,AB/1 (Item 1 from file: 340)

DIALOG(R)File 340:CLAIMS(R)/US Patent

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Dialog Acc No: 10632258

IFI Chemical Acc No: 2004-0040710

Document Type: C

CDNA ENCODING A GENE BOG (B5T OVER-EXPRESSED GENE) AND ITS PROTEIN PRODUCT;

TUMOR SUPPRESSION LIGAND COMPRISING RETINOBLASTOMA AND CASEIN BINDING

MOTIVES FOR USE TREATMENT OF CANCER; ANTITUMOR AGENTS

Inventors: Thorgeirsson Snorri S (US); Woitach Joseph T (US); Zhang

Minghuang (US)

Assignee: U S of America Health & Human Services

Assignee Code: 06814

Attorney, Agent or Firm: Attention: Katherine M. Kowalchyk;MERCHANT & GOULD

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Publication (No,Kind,Date), Applic (No,Date):

US 20040139485 A1 20040715 US 2004772988 20040205

Continuation Pub(No),Applic(No,Date): Pending

WO

99US4142 19990225

Division Pub(No),Applic(No,Date): US 6727079

US

2000637746 20000811

Priority Applic(No,Date): US 2004772988 20040205; WO 99US4142

19990225; US 2000637746 20000811

Provisional Applic(No,Date): US 60-75922 19980225; US 60-79567

19980327

The human papillomavirus E7 oncoprotein and the cellular transcription factor E2F bind to separate sites on the retinoblastoma tumor suppressor protein.

Wu E W; Clemens K E; Heck D V; Munger K

Laboratory of Tumor Virus Biology, National Cancer Institute, Bethesda, Maryland 20892.

Journal of virology (UNITED STATES) Apr 1993, 67 (4) p2402-7, ISSN 0022-538X--Print Journal Code: 0113724

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The ability of the high-risk and low-risk human papillomavirus E7 oncoproteins to disrupt complexes of the retinoblastoma tumor suppressor protein pRB and the cellular transcription factor E2F was studied. The ability of E7 to disrupt this transcription factor complex correlated with the different pRB binding efficiencies of the high-risk and low-risk human papillomavirus-encoded E7 proteins. The ***pRB*** ***binding*** site was the sole determinant for these observed differences. The phosphorylation status of the casein kinase II site that is immediately adjacent to the pRB binding site in E7 had no marked effect on this biochemical property of E7. Peptides consisting of the pRB binding site of E7, however, were not able to disrupt the pRB/E2F complex. These data suggest that additional carboxy-terminal sequences in E7 are also required for the efficient disruption of the pRB/E2F complex and that E7 and E2F may interact with nonidentical sites of pRB.

Analysis of the interaction between human papillomavirus type 16 E7 and the TATA-binding protein, TBP.

Phillips A C; Vousden K H

ABL Basic Research Program, NCI-FCRDC, Frederick, MD 21701-1201, USA.

Journal of general virology (ENGLAND) Apr 1997, 78 (Pt 4) p905-9,

ISSN 0022-1317--Print Journal Code: 0077340

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The E7 protein encoded by human papillomavirus type 16 shows transforming and immortalizing activities which are mediated, in part, through the interaction of the viral oncoprotein with the pRB protein family. This interaction is not solely responsible for E7 function, however, and other properties of E7, such as the interaction with basal transcription factors such as TBP, are likely to be of importance. We show here that three regions of the viral protein contribute to the interaction between E7 and TBP; the pRB-binding domain, the casein kinase II phosphorylation region and the C-terminal dimerization domain. Mutations within each region reduced the interaction of E7 with TBP in vitro, and simultaneous alterations within each of these regions completely abrogated binding. Unlike the pRB interaction, the association of E7 with TBP was enhanced following phosphorylation of E7 by casein kinase II, demonstrating a functional significance for ***phosphorylation*** of the viral protein.

4/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11580832 PMID: 9426450

Destabilization of the RB tumor suppressor protein and stabilization of p53 contribute to HPV type 16 E7-induced apoptosis.

Jones D L; Thompson D A; Munger K

Program in Biological and Biomedical Sciences, Harvard Medical School, Boston, Massachusetts 02115, USA.

Virology (UNITED STATES) Dec 8 1997, 239 (1) p97-107, ISSN 0042-6822--Print Journal Code: 0110674

Contract/Grant No.: CA 66980; CA; NCI; ES 07155; ES; NIEHS

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Cells that express the human papillomavirus (HPV) type 16 E7 oncoprotein are predisposed to undergo apoptosis. Transgenic mice that have E7 expression targeted to either the retinal photoreceptor cells or the lens cells exhibit signs of apoptosis in cells attempting to undergo differentiation. We established a cell culture system to study this process and have determined the domains of E7 that are required for predisposing cells to undergo apoptosis in response to growth arrest signals. Regions within the core pRB binding site of E7 were necessary but not sufficient for inducing apoptosis. Residues within the adenovirus conserved region 1 homology domain and the consensus casein kinase II phosphorylation site are also important for this effect on cell viability. Our data also demonstrate that the ability of E7 to induce destabilization of pRB and stabilization of p53 coincides with E7-mediated transformation and apoptosis.

... predisposing cells to undergo apoptosis in response to growth arrest signals. Regions within the core ***pRB*** ***binding*** site of E7 were necessary but not sufficient for inducing apoptosis. Residues within the

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Set	Items	Description
---	-----	-----
? s prb(w)binding		
	10062	PRB
	2166351	BINDING
S1	424	PRB(W)BINDING
? s casein(w)kinase(w)II(5n)phosphorylat?		
	70955	CASEIN
	866055	KINASE
	2053110	II
	461298	PHOSPHORYLAT?
S2	2725	CASEIN(W) KINASE(W) II (5N) PHOSPHORYLAT?
? s s1 and s2		
	424	S1
	2725	S2
S3	19	S1 AND S2